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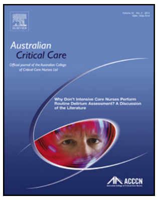

Evaluation of stress response using psychological, biological, and electrophysiological markers during immersive simulation of life threatening events in multidisciplinary teams

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ABSTRACT

Stress might impair clinical performance in real life and in simulation-based education (SBE). Subjective or objective measures can be used to assess stress during SBE. This monocentric study aimed to evaluate the effects of simulation of life-threatening events on measurements of various stress parameters (psychological, biological, and electrophysiological parameters) in multidisciplinary teams (MDTs) during SBE. The effect of gender and status of participants on stress response was also investigated. Twelve emergency MDTs of 4 individuals were recruited for an immersive simulation session. Stress was assessed by: (1) self-reported stress; (2) Holter analysis, including heart rate and heart rate variability in the temporal and spectral domain (autonomic nervous system); (3) salivary cortisol (hypothalamic pituitary adrenal axis). Forty-eight participants (54.2% men, <7years of experience) were included. Measures were performed at baseline (T0), after simulation (T1), after debriefing (T2), and 30 min after end of debriefing (T3). There was an increase in stress level at T1 ($p < 0.001$) and a decrease at T2 ($p < 0.001$). However, the variations of stress parameters induced by simulation (T0–T1 difference and T1–T2 difference) estimated by the three approaches were not correlated, while, as expected, Holter parameters were well-correlated to each other. Immersive SBE produced a change of stress level in all MDT members with no evidence for status effect but with gender difference. None developed a PTSD. These results support the hypothesis of a complementarity of the stress paths (collective reaction with increased stress level during simulation and a decrease during debriefing) but with relative independence of these paths (lack of correlation to each other). This study also suggests that because of the lack of correlation, stress response should be assessed by a combination of psychological, biological and electrophysiological parameters.

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1. Introduction

Stress is defined as a state of real or perceived threat to homeostasis.[1] It requires activation of a complex range of responses involving the endocrine, nervous, and immune systems, collec-

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tively known as the stress response. Stress is both a psychological and a physiological phenomenon[2] and the physiological stress response to the stimuli is accompanied by an anxiety state (psychological stress). Excessive stress can lead to post-traumatic stress disorder (PTSD).[3] In health care activities workload, leadership issues, professional conflicts, and emotional care demands combine to generate stress.[4] This stress impacts performance, described by an inverted U-shaped curve in animal and human studies,[5,6] and contributes to the definition of the arousal theory.[7] Excessive stress and poor management can lead to human errors and decrease recognition of these errors in real life and in simulation-based education (SBE)[8] independently of professional experience.[9] The effect of stress on a medical team during emergency management of patients requires consideration, since it can compromise the safety of patients.[10] Moreover, excessive stress interferes with the retrieval of conceptual knowledge and impedes problem solving.[11] Therefore, evaluation of stress during SBE is of interest to enhance the pedagogical impact of SBE. While debriefing appears to be a crucial time in SBE,[12,13] it is important to assess stress response during the simulation as well. Acute stress activates the autonomic nervous system (ANS), as can be apprehended by the acceleration of heart rate (HR) and modification in its variability (HRV) on Holter.[14,15] Stress also activates the Hypothalamic Pituitary Adrenal (HPA) axis activity, inducing biologic modifications such as an increase in cortisol that can be assessed by salivary cortisol (SC) in simulation.[16] Finally, some events can induce perceived-stress; and a subjective approach to assessment of stress is commonly used in simulation to measure its impact.[17] However, the relationship between self-reported and physiological stress is not unequivocal[2] and may even be controversial.[18] Consequently, stress response can be assessed by objective and/or subjective measures.[19] A combination of some biological, electrophysiological, and psychological measures of stress has been reported.[20] However, most studies have described the use of only one objective parameter of stress[18,21,22] or subjective parameter of stress.[23–26] Post-traumatic stress disorder (PTSD) can develop following a stressful event.[3] Although SBE can produce excessive stress, the occurrence of PTSD, relating to experiences in the simulated learning environment, has never been studied. PTSD usually occurs from one week to one month after a psychologically traumatic event.[27]

We hypothesized that all multidisciplinary team (MDT) members would experience stress during immersive SBE and that it would decline after debriefing. Because of their different physiological mechanisms, we did not expect a statistical correlation between subjective and objective stress measurements. We speculated that stress response would differ according to gender and status within a team with a higher stress level in the leader. We assumed there would be no PTSD among the participants after a simulation session.

The objective of this study was to evaluate the effects of simulation of life-threatening events on measurements of various stress parameters (electrophysiological, biological, and psychological parameters) in MDTs. Secondary objectives were: (1) to evaluate the correlation between the different stress parameters (2) to study stress response according to status and gender; and (3) to search for the occurrence of PTSD.

2. Methods

2.1. Study

This study took place in the ABS-Lab – Simulation Laboratory – INSERM (French national health and medical research institute) #1402, Faculty of Medicine of Poitiers, France. The study protocol, information form, and consent form were approved by the Comité de Protection des Personnes III de la region Ouest (Western France Person Protection Committee III) and were registered under the number 13.05.16. This study is the first part of a randomized single-center study on the relationship between stress and performance, registered by ClinicalTrials.gov under the number NCT02424890. All details of study protocol were reported previously.[28] The calculation of the number of subjects was based on an expected correlation of 0.50 between stress and performance. Four random samples of 12 participants for each of the 4 statuses present in an EMS team [Emergency Physician (EP), Residents (PGY i.e. Post Graduate Year), Registered Nurse (RN), and Ambulance Driver (AD)] were obtained. Each of the 12 MDTs was randomly constituted.

2.2. Population

All participants had less than 7 years of experience working in an EMS. This same level of experience allowed comparison in stress parameters between MDT members since stress depends on the competence and capacity of MDT leadership and co-workers.[29] EPs worked in one of the hospitals in the Poitou-Charentes region (1.8 10^6 inhabitants). They had obtained the University Diploma of Pediatric Emergency Procedures (University of Poitiers, France) after issuance of the American Heart Association and European Resuscitation Council guidelines in 2010.[30] During the university course they had received identical training in pediatric insertion of intraosseous (IO) access with performance assessment on the validated scale for simulated IO insertion.[31] PGYs specialized in Emergency Medicine, were trained in pediatric emergency procedures: clinical training in a Pediatric Emergency Unit and/or the university course. All health care providers (RNs, ADs) from the EMS of the University Hospital of Poitiers had obtained the European Pediatric Immediate Life Support degree over the two years preceding the session. All these EMS teams manage pediatric or adult patients in clinical practice.

2.3. Intervention

A high-fidelity manikin (SimNewB*, Laerdal®) was used for immersive simulation of an infant with hypovolemic shock requiring IO access (Supplemental file 1). All sessions were scheduled in a simulated resuscitation room (Supplemental file 2) on the same day of the week at 2:00pm because of the cortisol circadian cycle. Each simulation – lasting 25–30 min – was preceded by briefing (15 min), and followed by debriefing (30–45 min). In addition, a 45–60 min "snack break" lasting until 5:00pm was planned after each simulation session to allow physiological variables to return to normal conditions. Stressful conditions were related to different sources:[28] the scenario itself, a realistic environment, and the untimely intrusion of simulated parents in the resuscitation room. Four periods of 15 min were dedicated to saliva sampling and to filling out the STAI questionnaire. The saliva sampling was done at the same time for all participants. The chosen period for electrophysiological analysis was 20 min long, starting from 10 minutes prior to the saliva sampling time, to 10 min after the saliva sampling time.

2.4. Measurement of stress parameters

2.4.1. Psychological parameters of acute stress and PTSD

The STAI — State-Trait Anxiety Inventory scale[32] is widely used in simulation.[18] STAI included 20 questions with a 4-point Likert scale. Scores ranged from 20 to 80, with an acute anxiety response to stress for a score over 48 and a normal score around 34/80.[33] Self-assessment of stress can also be evaluated by the Stress-O-Meter (SOM) scale, with a score ranging from 0 to 10.[17,34] The occurrence of PTSD can be assessed using the Impact of Event Scale-Revised (IES-R) on the 7th day after the event[35] and the Post-traumatic Check-List Scale (PCLS) after one month.[36] IES-R ranged from 0 to

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88 and PCLS from 17 to 85. PTSD was defined by IES-R ≥ 36/88[35] and PCLS ≥ 44/85.[36] Participants filled out questionnaires in writing (E-mail).

2.4.2. Holter parameters

Holter parameters (HR (bpm), pNN50 and the LF/HF ratio) were obtained with the Synscope® (Sorin Group®) software during 24 h of recording, starting on the day prior to the simulation until the break after simulation. Cardiovascular activity was assessed by HR and HRV through time domain with pNN50 (proportion of successive NN that differ by more than 50 ms divided by total number of NN)[37] and the fast Fourier transformation (FFT) to perform spectral analysis and calculate LF/HF ratio (low and high frequency) that represents an estimation of "sympathovagal balance".[38,39] The sympathetic and parasympathetic activity always interfere with HR. HRV which reflect this interaction provide a measure of the sympathetic and parasympathetic activity induced by the stress response.[40] It has been shown that acute stress, of which the existence is confirmed by an increase in SC, involves a decrease in pNN50[16] and an increase in LF/HF.[41]

2.4.3. Salivary cortisol

SC is a faithful reflection of biological stress, with the major advantage that it does not require invasive sampling techniques.[42] SC (μg/dl) was measured by an enzyme-linked immunosorbent assay (ELISA)[43] with an ELISA kit (IBL international®, Hamburg, Germany).[44] Different parameters such as diet, nicotine and exercise may affect salivary cortisol but their impact was limited. The participants did not engage in any exercise the day before the simulation. Moreover, as instructed by IBL procedure the participants did not eat, drink, chew gum, smoke, or brush their teeth during the 30 min before sampling saliva, or rinse their mouths 5 min before sampling.[44]

2.5. Statistical analysis

Quantitative variables were described as mean and standard deviation (M ± SD). Categorical variables were summarized by frequencies and percentages. Stress scores were normalized on 100 except for SOM, which was given on 10. The Kolmogorov-Smirnov test was used to check normal distribution for the measures assessed. Stress measurements were compared between baseline (T0) and the other times (T1, T2 and T3) using ANOVA for repeated measures (RM-ANOVA). Post hoc multiple comparisons among the groups were made using the Scheffe test. Regarding Stress-O-Meter comparison between T1 and T2 was performed using a pairwise student t test. Changes in stress level between 2 times were assessed by calculation of the relative variation ((final score − initial score)/initial score). Correlations between the stress parameters were evaluated with Spearman correlation coefficient. Stress responses were compared between socio-professional groups using a Kruskal–Wallis test and between males and females with a Mann–Whitney test. All tests were 2-sided, with type I error rate fixed at 0.05. All tests were performed using Statistical Analysis Software (SAS, version 9.3; SAS Institute, Cary, NC).

3. Results

3.1. Population

Forty-eight participants (54.2% males) from the 12 MDTs who participated in high-fidelity simulation of emergency treatment of an infant with hypovolemic shock were considered in the analysis. The paramedics (RNs and ADs) were older than the EPs and the PGYs (32 ± 9 for EPs, 29 ± 8 for PGYs, 42 ± 13 for RNs, and 47 ± 16 for ADs), and all of them had less than 7 years of EMS experience. The paramedics (RNs and ADs) had generally practiced in other departments before joining the EMS.

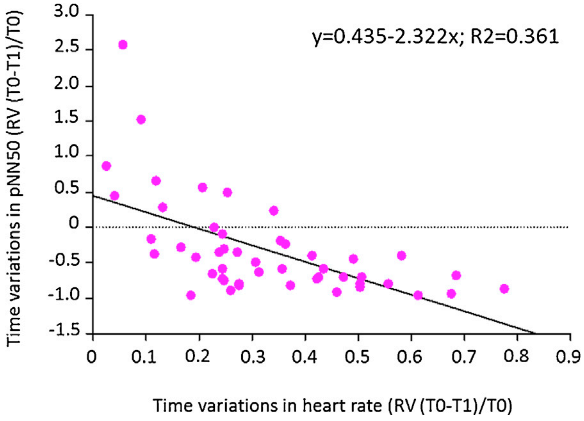

Fig. 1. Correlations between time-variations in heart rate and time-variations in heart rate variability estimated by time domain analysis (pNN50).
pNN50: percentage of RR′ intervals >500 ms; RV: Relative variation ((final score − initial score)/initial score); T0: baseline; T1: post-simulation.

3.2. Measurement of stress parameters (Table 1)

Stress was studied on 12 MDTs of French EMS insofar as it might impair clinical performance in real life and in SBE. During the simulation session stress level increased with a significant variation of stress measures between 'pre-simulation time' = baseline (T0) and 'immediately after simulation time' (T1): STAI, HR, LF/HF ratio, SC increased significantly between T0 and T1 whereas pNN50 decreased. 'Immediately after debriefing time' (T2) there was a significant decrease in the psychological and biological measures of stress, which all nonetheless remained above baseline (T0). HR was significantly lower and pNN50 significantly higher 30 min after debriefing time (T3) than immediately after. The SOM score significantly increased between T0 and T1. Results are given in Table 1.

3.3. Correlations between stress parameters (Table 2)

Globally, there was no correlation between the different methods regarding changes in stress parameters induced by simulation (T0–T1 difference, T1–T2 difference, and T0–T2 difference), with some subjects showing a more pronounced response in the psychological assessment, and others on the electrophysiological or biological parameters. The correlation between SC and STAI changes from T0 to T1 (rho = 0.33, p = 0.02), and between SC and LF/HF ratio changes from T0 to T2 (rho 0.41, p = 0.006) were the only significant ones. Regarding the electrophysiological parameters, as expected there was a strong correlation between HR and HRV (Figs. 1 and 2), and also between time domain analyses and spectral domain analyses of HRV.

3.4. Status and gender effects

Changes in STAI scores, Holter parameters, and salivary cortisol measurements of stress induced by simulation were independent of status. While under baseline condition (T0) there was no gender difference in stress level, the stress responses induced by simulation between male and female were not similar. During simulation females displayed greater psychological stress with more recovery response after debriefing than males. STAI at T1 was higher in females (62.5 ± 14.0 vs. 50.7 ± 10.7, p = 0.004). HR was significantly higher in females than in males at T1 (90.8 ± 12.2 vs. 81.3 ± 10.9,

Table 1
Objective and subjective stress parameters performed at T0, T1, T2, and T3, followed by between-time comparisons.

Measures(mean ± SD)	T0	T1	T2	T3	ANOVA F(3, 44)	Comparison T0 vs. T1	Comparison T1 vs. T2	Comparison T2 vs. T3
SC	0.27 (0.14)	0.36 (0.24)	0.25 (0.15)	ND	9.8 p = 0.001	p = 0.02	p = 0.007	NA
HR	76.0 (9.9)	85.8 (12.4)	85.7 (11.7)	74.5 (8.6)	91.7 p < 0.001	p < 0.001	p = 0.99	p < 0.001
pNN50	16.3 (15.9)	9.4 (8.4)	10.1 (9.5)	15.8 (12.9)	15.9 p < 0.001	p < 0.001	p = 0.98	p = 0.01
LF/HF	4.5 (2.8)	6.3 (3.4)	6.1 (3.1)	5.9 (3.7)	19.6 p < 0.001	p < 0.001	p = 0.94	p = 0.99
STAI	48.3 (12.2)	56.4 (16.6)	40.6 (7.9)	ND	35.8 p < 0.001	p < 0.001	p < 0.001	NA
SOM	ND	5.2 (2.1)	7.2 (1.8)	ND	NA	NA	t(47) = −5.98 p < 0.001	NA

ANS: autonomic nervous system; HF: high frequency (Hz); HPA: hypothalamic–pituitary–adrenal axis; HR: heart rate (bpm); LF: low frequency (Hz); NA: not applicable; pNN50: proportion of successive NN that differ by more than 50 ms divided by total number of NN; SC: salivary cortisol (μg/dl); SOM: Stress-O-Meter (0–10); STAI: State-Trait Anxiety Inventory scale (over 100); T0: baseline; T1: post-simulation; T2: post-debriefing; T3: 30 min after debriefing time; ND: not evaluated; Values are means (standard deviations); Between-time comparisons were done by ANOVA for repeated measures and scheffé test except for SOM compared by t-test for paired measurements.

Table 2
Correlations between stress parameters.

Correlation	Between T0 and T1		Between T1 and T2		Between T0 and T2	
	Rho	p	Rho	p	Rho	p
RV HR/RV pNN50	−0,64	**<0,0001**	−0,62	**<0,0001**	−0,74	**<0,0001**
RV HR/RV LF/HF	0,01	0,96	0,20	0,17	0,38	**0,01**
RV HR/RV SC	−0,08	0,60	−0,13	0,37	0,01	0,93
RV HR/RV STAI	0,25	0,09	−0,08	0,59	−0,03	0,86
RV HR/RV SOM	−0,15	0,30	NA	NA	NA	NA
RV pNN50/RV LF/HF	−0,38	**0,009**	−0,56	**0,0002**	−0,50	**0,0008**
RV pNN50/RV SC	0,06	0,67	0,03	0,85	−0,07	0,62
RV pNN50/RV STAI	−0,05	0,75	−0,10	0,51	−0,01	0,92
RV pNN50/RV SOM	−0,01	0,93	NA	NA	NA	NA
RV LF/HF/RV SC	0,21	0,16	0,01	0,98	0,41	**0,006**
RV LF/HF/RV STAI	0,10	0,51	0,11	0,47	0,06	0,38
RV LF/HF/RV SOM	−0,03	0,85	NA	NA	NA	NA
RV SC/VR STAI	0,33	**0,02**	0,29	0,05	−0,12	0,31
RV SC/RV SOM	−0,01	0,94	NA	NA	NA	NA
RV STAI/RV SOM	0,13	0,37	NA	NA	NA	NA

HF: high frequency (Hz); HR: heart rate (bpm); LF: low frequency (Hz); NA: not applicable; pNN50: proportion of successive NN that differ by more than 50 ms divided by total number of NN; RV: relative variation ((final score — initial score)/initial score); Rho: Spearman correlation coefficient; SC: salivary cortisol (μg/dl); SOM: Stress-O-Meter (0–10); STAI: State-Trait Anxiety Inventory scale (over 100); T0: baseline; T1: post-simulation; T2: post-debriefing.

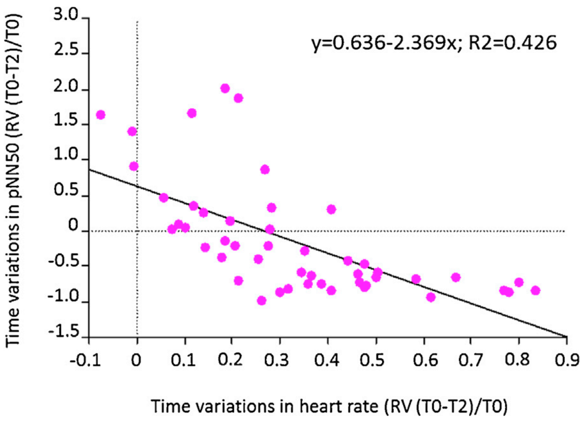

Fig. 2. Correlations between time-variations in heart rate and time-variations in heart rate variability estimated by time domain analysis (pNN50).
pNN50: percentage of RR′ intervals >500 ms; RV: Relative variation ((final score — initial score)/initial score); T0: baseline; T2: post-debriefing.

$p = 0.005$) and T2 (89.4 ± 9.7 vs. 82.3 ± 12.5, $p = 0.03$). LF/HF ratio was lower in females than in males (4.70 ± 2.31 vs. 7.25 ± 3.18, $p = 0.007$). After debriefing STAI and LF/HF ratio decreased with more rapid recovery in females, whereas SC decreased with more rapid recovery in males. Relative variation of STAI between T1 (post-simulation) and T2 (post-debriefing) was -0.18 ± 0.18 for males vs. -0.32 ± 0.19 for females ($p = 0.02$). Relative variation of LF/HF ratio between T1 and T2 was 0.34 ($SD = 0.74$) for males vs. -0.09 ($SD = 0.45$) for females ($p = 0.03$). Finally, relative variation of SC between T1 and T2 was -0.31 ± 0.17 for males vs. -0.21 ± 0.15 for females ($p = 0.03$).

3.5. *Post-traumatic stress disorder*

No participant developed PTSD. Scores were transformed over 100. IES-R score was 11.7 ± 1.9 with a maximum at 34 and PCLS score was 19.0 ± 0.8 with a maximum at 29.

4. Discussion

4.1. *Main results*

The aim of this study was to evaluate the effects of simulation of management of life-threatening events on measurements of various stress parameters, pertaining to the impairment of performance by stress. In a simulation session with a model of infant

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life-threatening emergency, stress level increased after simulation and decreased after debriefing for the 48 MDT care providers. STAI and SC measures significantly decreased immediately after debriefing, whereas the Holter parameters decreased later in the temporal domain but not in the spectral domain. There was no correlation between changes of the various stress parameters over time except for the Holter parameters between each other. There was no status effect. Females displayed a higher stress response during the simulation with a more rapid recovery for psychological and electrophysiological parameters. However, males exhibited a more rapid recovery in cortisol level. Although the simulations were stressful, there was no PTSD. To our knowledge, no previous study had searched for occurrence of PTSD during SBE.

4.2. Primary objective

The present study evidenced a rise in all parameters of stress during simulation of MDT management of an infant in shock. Similar results were found in residents for perceived stress (Likert scale as SOM), HR and HRV, but not for blood cortisol after resuscitation simulation.[45] The strength of the present study is to have studied stress response with multiple components as suggested by Laurent[46] in high-fidelity simulation: perceived stress by 2 components (STAI and SOM) and objective measures of stress by Holter parameters and SC. Arora et al. suggested a similar approach to study stress response in the operating room. The authors proposed the use of the Imperial Stress Assessment Tool (ISAT), which is a combination of STAI, SC, and HR.[47] SC has the advantage of being a non-invasive method compared to the plasma cortisol determination used in other studies.[45] Holter parameters included complete analysis of electrophysiological parameters (HR, HRV in time and frequency domains).[19] Few studies have investigated these two axes in a clinical scene[46] or in SBE.[18] Furthermore, they were lacking in a standardized measure of stress.[18,20] In the literature, usually when several measures of stress are employed they reflect only one pathway[48] or the association of an objective measure with self-report.[49] In SBE studies all stress pathways need to be assessed because variation in the systems could be different.[50] Moreover, it would be interesting to estimate the task level for participants and thereby predict excessive stress during training.[51] The present study showed an activation of all stress axes during the simulation session, with a decline occurring after debriefing. Decrease in stress level after debriefing has never been described. This could be related to the good-judgement debriefing procedure per se.[52] But in the absence of a control group (without debriefing), spontaneous decrease in stress measures after simulation cannot conclusively be ruled out. After debriefing, the decrease in SC and perceived stress was faster than for Holter parameters. Moreover, while the spectral domain parameters remained stable, there was a decrease in stress parameters calculated in the temporal domain. These results suggest a more complex relationship between stress, HR and its different estimated variability parameters (temporal and frequency/spectral), and the evidence of LF/HF variation such as sympathetic modulation[53] and parasympathetic system withdrawal.[54] Nevertheless, the present results reinforce the idea that scheme of frequency bands cannot fully represent a model of ANS response to stressors,[55] as multiple factors are liable to interfere with sympathovagal balance.

4.3. Secondary objectives

Despite all stress markers increasing during simulation and declining after debriefing, there was no correlation (except for HR and HRV) between their absolute values and their variations. However, for ANS parameters there was a higher correlation between HR and pNN50 (i.e. temporal domain analysis) than HR and LF/HF ratio (i.e. spectral domain analysis). Our results suggested that the use of LF/HF ratio as a surrogate for sympathovagal balance was contentious. The LF/HF ratio may not accurately measure cardiac sympathovagal balance, particularly because of the poor relationship between LF power and sympathetic nerve activation described in the litterature.[56,57] Surprisingly, Hunziker found a correlation between plasma cortisol, HR, HRV, and perceived stress on baseline that contrasted with the absence of correlation after simulation and debriefing except for HR and HRV.[45] Variations of objective measures of stress differed between each other in two dimensions: cortisol rate of change differed from that of electrophysiological measures and variation over time was not similar. Higher perceived stress during simulation was not correlated with ‘biologic stress parameters’. This study seemed to evidence that the link between stress pathways is not so simple. On the contrary, the mechanisms of biological – objective – and subjective stress parameters expression are complex, as is suggested by contradictory results.[2,16,19,23,33] Absence of correlation might be explained by differences between stress response circuits. Stress response is regulated by neural circuits impinging on the Hypothalamic Pituitary Adrenal (HPA) stress axis[1] and the sympathetic branch of the ANS.[58] ANS leads to a rapid response for coping mechanisms by modulation of electrophysiological parameters and released catecholamines whereas cortisol is issued from a slower cascade activated by HPA.[46] Cortisol levels peak at 30–40 min after the onset of stressors.[59] Indeed, even if HPA and ANS are interrelated and interact,[60] there was no correlation in spite of a non-equivocal increase in all measures of stress during simulation. Some authors have described ANS parameters as reflecting an ‘effort or challenge’ (alertness and action proneness) component of stress and HPA cascade and a ‘distress’ (negative feeling) component.[46] One explanation of the absence of observed correlation between objective and subjective measures of stress parameters could be the interplay of the neurohormonal axis, circadian rhythms, and individual variability in chronotropic response to stress.[61] Moreover, Metzenthin et al. found that concurrently self-reported stress like SOM was related to SC but that this was not the case if it was retrospectively determined.[49] In short, face with a lack of correlation between stress parameters it may be advisable to investigate parameters off all pathways, and also to pay particular attention to subjective parameters when investigating stress response in simulation courses.

The present study found that as previously described,[18,62] all participants exhibited a similar stress response irrespective of their status. This suggests that stress might be assessed in all team members and rather than focusing on one of them (leader). Indeed, excessive stress in one team member may impair teamwork.[63] As expected, there were gender differences according to stress response. However, females expressed a higher autonomic response on baseline than males, whereas usually, stress is higher in males.[64,65] But other authors have suggested that there is no gender difference or that stress response could be greater in females.[59,66] Similarly and as previously suggested,[67] females expressed more anxiety during simulation. Analysis of relative variation of SC indicated that SC decreased more quickly among males after debriefing. McGraw et al. suggested larger changes in SC level in males and more rapid recovery after simulation.[68] However, to our knowledge, this variation after debriefing has never been described. Conversely, decrease of STAI after debriefing was more pronounced in females than in males. Finally, relative variation of LF/HF ratio varied in the opposite way in females and males reinforcing the hypothesis that multiple factors might interfere with sympathovagal balance and gender difference. All in all, the heterogeneity of these results highlighted the complex nature of gender-specific stress response.[69] Our results showed that gender

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could be treated as an independent variable in all mixed-gender studies in simulation. To our knowledge this is the first simulation study addressing the ECG signal by spectral analysis (LF/HF), thereby allowing a more thorough approach to stress.[28]

In the present study, no participant developed PTSD after the experiment. PTSD has been described in individuals exposed to exceptional events during emergency medicine practice with an immediate acute stress response.[70] To our knowledge, no simulation study has sought out an occurrence of PTSD in the SBE environment. We supposed that using standardized debriefing with 'good-judgement method'[52] might contribute to the absence of PTSD. Moreover, due to our Simulation Center policy regarding death no simulated patient died. Indeed, emotional reactions to simulated death can have a serious psychological impact, particularly when the death results from actions or inactions by the learner.[71] The non-occurrence of death of the simulated patient in our experiments may have helped to limit risk of PTSD.

4.4. Limitations

Despite the selection of participants and the strict protocol, this study had some limitations. Paramedics were older than EPs and PGYs. This difference should not be a bias since age does not seem to interfere in stress mechanisms beyond adolescence[72] whereas level of experience should interfere in stress response.[68] All participants were filmed and observed, which may have increased their stress level.[73] This effect was nonetheless limited by a safe environment[74] with a briefing explaining the presence of the observers. The participants were also reminded that during debriefing there was no person in judgement. On the other hand, some participants said they were bothered by the electrodes set up to record Holter parameters, which may have modified their level of stress. However, this factor would not have changed the relative variation of stress parameters allowing for study of stress response.

5. Conclusion

The present high-fidelity simulation scenario tested on twelve multidisciplinary teams was a source of stress for all team members. As suspected, we found a gender difference in stress response. There was no correlation between STAI, Holter parameters, and salivary cortisol, thereby reinforcing the hypothesis of complex stress physiology. This study strongly favours the use of combined objective and subjective measures of stress to investigate stress response in simulation-based education. Moreover, use of such measures might include components of the Hypothalamic Pituitary Adrenal axis and the Autonomic Nervous System. Gender should be treated as an independent variable in all mixed-gender studies involving high-fidelity simulation. Stress is an important component associated with simulation-based education designed to approach real-life conditions in emergency situations by creating a realistic learning environment. Future studies should investigate the impact of stress response on performance. Simulation represents a useful and effective means of promoting and advancing best practice in critical situations. Stress assessment could help to estimate difficult levels of tasks and training could enhance participant performance under stressful conditions.

Ethics

This study took place in the ABS-Lab – Simulation Laboratory – INSERM (French national health and medical research institute) #1402, Faculty of Medicine of Poitiers, France. The study protocol, information form, and consent form were approved by the Comité de Protection des Personnes III de la region Ouest (Western France Person Protection Committee III) and were registered under the number 13.05.16. This study is the first part of a randomized single-center study on the relationship between stress and performance, registered by ClinicalTrials.gov under the number NCT02424890.

Authors' contributions

Daniel Aiham GHAZALI, MD, PhD, made substantial contributions to conception and design, performed acquisition of data, data management, and drafted the manuscript.

Ivan DARMIAN-RAFEI, MD, made substantial contributions to acquisition of psychological questionnaires.

Jerome NADOLNY, RN, made substantial contributions to acquisition of biological parameters.

Philippe SOSNER, MD, PhD, performed analysis of Holter parameters.

Stephanie RAGOT, MD, PhD, made substantial contributions to conception and design, performed statistical analysis of data, and was involved in drafting the manuscript.

Denis ORIOT, MD, PhD, made substantial contributions to conception and design, provided substantial help for interpretation of data, and reviewed the manuscript.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.aucc.2017.07.001.

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